We Claim:

1. A method of assessing cancer in a body sample of a human suspected of havng a cancer, comprising the steps of:

   determining a non-synonymous, intragenic mutation in a PIK3CA coding sequence in the body sample, wherein a wild-type PIK3CA coding sequence comprises the sequence shown in SEQ ID NO:2;

   identifying the human as likely to have cancer if a non-synonymous, intragenic mutation in PIK3CA coding sequence is determined in the body sample.

2. The method of claim 1 wherein the body sample is a first tissue that is suspected of being neoplastic, and the method further comprises the steps of:

   testing a second tissue that is not suspected of being neoplastic for the presence of the non-synonymous mutation, wherein the first and second tissue are isolated from the human;

   identifying the non-synonymous, intragenic mutation as somatic if said mutation is absent in the second tissue.

3. The method of claim 1 wherein the non-synonymous, intragenic mutation is in exon 9 (SEQ ID NO: 4).
4. The method of claim 1 wherein the non-synonymous, intragenic mutation is in exon 20 (SEQ ID NO: 5).
5. The method of claim 1 wherein the non-synonymous, intragenic mutation is in PIK3CA's helical domain (nt 1567-2124 of SEQ ID NO: 2).
6. The method of claim 1 wherein the non-synonymous, intragenic mutation is in PIK3CA's kinase domain (nt 2095-3096 of SEQ ID NO: 2).
7. The method of claim 1 wherein the non-synonymous, intragenic mutation is in PIK3CA's P85BD domain (nt 103-335 of SEQ ID NO: 2).
8. The method of claim 1 wherein the body sample is colorectal tissue.
9. The method of claim 1 wherein the body sample is brain tissue.
10. The method of claim 1 wherein the body sample is gastric tissue.
11. The method of claim 1 wherein the body sample is breast tissue.

12. The method of claim 1 wherein the body sample is lung tissue.
13. The method of claim 1 wherein the body sample is blood, serum, or plasma.
14. The method of claim 1 wherein the body sample is sputum.
15. The method of claim 1 wherein the body sample is saliva.
16. The method of claim 1 wherein the body sample is urine.
17. The method of claim 1 wherein the body sample is stool.
18. The method of claim 1 wherein the body sample is nipple aspirate.
19. The method of claim 1 wherein PIK3CA exons consisting of 9 and 20 are tested to determine a non-synonymous mutation.
20. The method of claim 1 wherein PIK3CA exons comprising 9 and 20 are tested to determine a non-synonymous mutation.
21. The method of claim 1 wherein the non-synonymous, intragenic mutation is a substitution mutation.
22. The method of claim 1 wherein the non-synonymous, intragenic mutation is G1624A.
23. The method of claim 1 wherein the non-synonymous, intragenic mutation is G1633A.
24. The method of claim 1 wherein the non-synonymous, intragenic mutation is C1636A.
25. The method of claim 1 wherein the non-synonymous, intragenic mutation is A3140G.
26. The method of claim 1 wherein the body sample is tested for mutations at nucleotide positions 1624, 1633, 1636, and 3140 of PIK3CA coding sequence.
27. The method of claim 1 wherein the body sample is tested for mutations G1624A, G1633A, C1636A, and A3140G.
28. The method of claim 21 wherein the body sample is further tested for mutations G113A, T1258C, G3129T, and C3139T.
29. The method of claim 27 wherein the body sample is further tested for mutation G2702T.
30. The method of claim 1 wherein the non-synonymous, intragenic mutation is a deletion mutation.
31. A method of inhibiting progression of a tumor in a human, comprising the steps of:

    administering to the human an antisense oligonucleotide or antisense construct to a tumor, wherein the antisense oligonucleotide or RNA transcribed from the antisense construct is complementary to mRNA transcribed from PIK3CA

(SEQ ID NO: 2), whereby amount of p110α protein expressed by the tumor is reduced.

32. The method of claim 31 wherein the antisense oligonucleotide or RNA transcribed from the antisense construct are complementary to a region of said mRNA which comprises an initial methionine codon of said mRNA.

33. A method of inhibiting progression of a tumor in a human, comprising the steps of:

administering to the human siRNA comprising 19 to 21 bp duplexes of a human PIK3CA mRNA with 2 nt 3' overhangs, wherein one strand of the duplex comprises a contiguous sequence selected from mRNA transcribed from PIK3CA (SEQ ID NO: 2), whereby amount of p110α protein expressed by the tumor is reduced.

34. The method of claim 33 wherein the contiguous sequence comprises an initial methionine codon of said mRNA.

35. A method of inhibiting progression of a tumor, comprising the steps of:

administering a molecule comprising an antibody binding region to a tumor, wherein the antibody binding region specifically binds to p110α (SEQ ID NO: 3).

36. The method of claim 35 wherein the antibody binding region specifically binds to the kinase domain (nt 2095-3096 of SEQ ID NO: 2) of PIK3CA.

37. The method of claim 35 wherein the antibody binding region specifically binds to the helical domain (nt 1567-2124 of SEQ ID NO: 2) of PIK3CA.

38. The method of claim 35 wherein the antibody binding region specifically binds to the P85BD domain (nt 103-335 of SEQ ID NO: 2) of PIK3CA.

39. A method of identifying candidate chemotherapeutic agents, comprising the steps of:

contacting a wild-type or activated mutant p110α (SEQ ID NO: 3) with a test compound;

measuring p110α activity;

identifying a test compound as a candidate chemotherapeutic agent if it inhibits p110α activity.

40. The method of claim 39 wherein a mutant form of the p110α is contacted with the test compound, said mutant form comprising a substitution mutation selected from the group consisting of E542K, E545K, Q546K, and H1047R.
41. The method of claim 39 wherein the test compound is identified as being a candidate chemotherapeutic agent for treating tumors with an activating mutation of p110α .
42. The method of claim 39 wherein the test compound is identified as being a candidate chemotherapeutic agent for treating tumors with a substitution mutation of PIK3CA.
43. The method of claim 39 wherein the test compound is identified as being a candidate chemotherapeutic agent for treating tumors with an activating mutation of PIK3CA in its kinase domain (nt 2095-3096 of SEQ ID NO: 2).
44. The method of claim 39 wherein the test compound is identified as being a candidate chemotherapeutic agent for treating tumors with an activating mutation of PIK3CA in its helical domain (nt 1567-2124 of SEQ ID NO: 2).
45. The method of claim 39 wherein the test compound is identified as being a candidate chemotherapeutic agent for treating tumors with an activating mutation of PIK3CA in its P85BD domain (nt 103-335 of SEQ ID NO: 2).
46. The method of claim 39 further comprising the steps of:

    contacting the test compound with one or more enzymes selected from the group consisting of: PIK3CB, PIK3CG, PIK3C2A, PIK3C2B, PIK3C2G, PIK3C3, A-TM, ATR, FRAP1, LAT1-3TM,SMG1, PRKDC, and TRRAP;

    identifying a test compound as a specific candidate chemotherapeutic agent if it inhibits one or more of said enzymes less than it inhibits p110α .
47. The method of claim 46 wherein a test compound which inhibits PIK3CB, PIK3CG, PIK3C2A, PIK3C2B, PIK3C2G, and PIK3C3 less than it inhibits p110α (PIK3CA) is identified as a highly specific candidate chemotherapeutic agent.
48. The method of claim 46 wherein a test compound which inhibits p110α more than it inhibits PIK3CB and PIK3CG is identified as highly specific.
49. The method of claim 39 wherein the step of contacting is performed in a cell-free system.
50. The method of claim 39 wherein the step of contacting is performed in whole cells.

51. A method for delivering an appropriate chemotherapeutic drug to a patient in need thereof, comprising:

determining a non-synonymous, intragenic mutation in a PIK3CA coding sequence (SEQ ID NO: 1) in a body sample of a patient;

administering a p110α inhibitor to the patient.

52. The method of claim 51 wherein the p110α inhibitor is LY294002.

53. The method of claim 51 wherein the p110α inhibitor is wortmannin.

54. The method of claim 51 wherein the p110α inhibitor is a molecule comprising an antibody binding region specific for p110α.

55. The method of claim 54 wherein the antibody binding region binds to the kinase domain (nt 2095-3096 of SEQ ID NO: 2).

56. The method of claim 54 wherein the antibody binding region binds to the helical domain (nt 1567-2124 of SEQ ID NO: 2).

57. The method of claim 54 wherein the antibody binding region binds to the P85BD domain (nt 103-335 of SEQ ID NO: 2).

58. A set of one or more primers for amplifying and/or sequencing PIK3CA, said primers selected from the group consisting of forward primers, reverse primers and sequencing primers, wherein the forward primers are selected from the group consisting of: SEQ ID NO: 6 to 165, the reverse primers are selected from the group consisting of: SEQ ID NO: 166 to 325, and the sequencing primers are selected from the group consisting of: SEQ ID NO: 326 to 485.

59. The set of claim 58 wherein the one or more primers comprise at least one forward and one reverse primer for amplifying a segment of PIK3CA.

60. The set of claim 58 wherein the one or more primers comprise at least one forward, one reverse, and one sequencing primer for amplifying and sequencing a segment of PIK3CA.

61. The set of claim 58 wherein the one or more primers comprise all of said forward, reverse, and sequencing primers.

62. The set of claim 58 which is in a single divided or undivided container.

63. The set of claim 59 which is in a single divided or undivided container.

64. The set of claim 60 which is in a single divided or undivided container.

65. The set of claim 61 which is in a single divided or undivided container.